Published in final edited form as:
J Child Psychol Psychiatry. 2012 March ; 53(3): 234–242. doi:10.1111/j.1469-7610.2011.02445.x.

A longitudinal twin study on the association between ADHD symptoms and reading

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Abstract

Background—Attention deficit hyperactivity disorder (ADHD) and reading disability commonly co-occur due to shared genetic risk factors. However, the stability and change of these genetic influences and the predictive relationships underlying this association longitudinally remain unclear.

Methods—ADHD symptoms and reading were assessed as continuous dimensions in a U.K. general population sample of approximately 7000 twin pairs. Parent ratings of ADHD symptoms and teacher ratings of reading were obtained at two ages: middle childhood (child ages 7–8) and early adolescence (child ages 11–12). Cross-lagged quantitative genetic analyses were applied.

Results—ADHD symptoms and reading significantly predicted each other over time. However, ADHD symptoms were a significantly stronger predictor of reading than vice versa. Inattentive and hyperactive-impulsive symptoms of ADHD both contributed to the prediction of reading, but inattentiveness was a significantly stronger predictor. Furthermore, ADHD symptoms and reading were highly heritable, and their association was primarily attributable to shared genetic influences. Despite notable genetic innovation for each trait, genetic factors involved in the *association* of ADHD symptoms and reading over time were highly stable.

Conclusions—ADHD symptoms may put children at increased risk for reading problems and vice versa. Moreover enduring genetic mechanisms appear to be important in the association of ADHD symptoms and reading over time.

Keywords

ADHD; inattentiveness; hyperactivity-impulsivity; reading; longitudinal; twin; genetics

Children with ADHD often experience reading disability. ADHD and reading disability co-occur more often than expected by chance, and in clinical and non-clinical samples (Semrud-Clikeman et al., 1992; Willcutt & Pennington, 2000), which suggests that their co-occurrence is not due to a sampling artefact.

Twin studies have shown that ADHD symptoms and reading are highly heritable (Faraone et al., 2005; Olson, 2006) and analysis of cross-sectional twin data suggests that their association is largely attributable to genetic influences shared between reading and inattentive symptoms of ADHD (Greven, Harlaar, Dale, & Plomin, 2011; Paloyelis,

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Conflict of interest: Asherson has acted in an advisory capacity to, and has given talks at meetings or educational programs sponsored by Shire, Janssen-Cilag, Eli-Lilly and Flynn Pharma. He is the holder of an educational grant from Janssen-Cilag and a research grant from Shire. The other authors have no competing interests.

Rijsdijk, Wood, Asherson, & Kuntsi, 2010; Willcutt, Pennington, Olson, & DeFries, 2007). However, although this cross-sectional evidence is highly consistent in demonstrating genetic overlap between ADHD symptoms and reading in samples including the childhood and adolescent ages, genetic influences on ADHD symptoms and reading are not perfectly stable (Harlaar, Dale, & Plomin, 2007; Kuntsi, Rijsdijk, Ronald, Asherson, & Plomin, 2005). This gives rise to the possibility that different genetic influences may be involved in genetic overlap between ADHD symptoms and reading over time. This is an important issue as it addresses whether the aetiologies of this association are stable or differ over time.

To date, only one twin study has examined the stability of genetic and environmental influences on the association of inattentiveness and reading (Ebejer et al., 2010). Based on a population-based sample of 989 twin pairs followed across ages 6–9, the study concluded that the genes involved in the association between inattentiveness and reading at study onset did not change with development. In the present study, we aim to replicate this finding in a substantially larger sample (around 7000 twin pairs), as well as to extend this finding to a period spanning middle childhood (ages 7–8) to early adolescence (ages 11–12).

The present study also sets out to examine the direction of effects underlying ADHD symptoms and reading over this period. It has previously been suggested that ADHD symptoms may lead to the development of reading disability, possibly because attention deficits interfere with reading instruction (e.g., Dally, 2006). On the other hand, reading disability may lead to the development of ADHD symptoms, possibly as a result of the academic failure experienced by the child (e.g., Cunningham, & Barkley, 1978).

Longitudinal studies of phenotypic (observed) data across childhood and adolescence have provided evidence for both directions of effects. For example, ADHD symptoms have been shown to predict later reading disability (e.g., Dally, 2006; McGee, Prior, Willams, Smart, & Sanson, 2002; Rabiner & Coie, 2000), and some evidence also suggests that reading disability predicts later ADHD symptoms (e.g., McGee et al., 2002; Rabiner et al., 2000).

An early influential study compared the neuropsychological profiles of children from three groups (ADHD only, reading disability only, and both conditions) and found that the neuropsychological profile of children from the comorbid group resembled the profile of children with reading disability only (Pennington, Groisser, & Welsh, 1993). Based on this, the study hypothesized that reading disability might be the primary disorder, which causes secondary symptoms of ADHD without the aetiology typically associated with ADHD. However, later neuropsychological studies using the same design did not replicate these findings (e.g., Reader, Harris, Schuerholz, & Denckla, 1994; Willcutt et al., 2001). Instead, several cognitive deficits, including processing and naming speed, were later shown to be shared between the disorders (e.g., Willcutt et al., 2010), in line with a common aetiology model.

Twin research has also helped address whether ADHD symptoms and reading may influence the development of each other. For example, a genetic association has been shown to exist already in pre-school children between inattentive symptoms and pre-reading skills (Willcutt, Betjemann et al., 2007), which suggests that the association between ADHD symptoms and reading is not attributable, at least not exclusively, to interference with formal reading instruction or academic frustration during the school years. Moreover the longitudinal twin study of ADHD symptoms and reading mentioned above (Ebejer et al., 2010) concluded that a common genes account best explains the relations between inattentiveness and reading at the end of kindergarten (around ages 6–7) and first grade (ages 7–8); however from second grade (ages 8–9 years) inattentiveness may begin to play a

more direct causal role in reading ability, possibly because cognitive demands to read successfully begin to expand around that age.

The present study

The present study extends previous research by examining, for the first time, the longitudinal relations between ADHD symptoms and reading from middle childhood (ages 7–8) to early adolescence (ages 11–12) in a quantitative genetic cross-lagged model. This model is well suited for examining the issues raised above, as it allows the examination of direction of causation and common aetiology hypotheses in a single model. Data came from an unselected population-representative sample of around 7000 twin pairs from the United Kingdom. Thus, rather than focusing on the clinical extreme, the present study examines the full range of the normal distribution of ADHD symptoms and reading ability.

Aims and hypotheses

The first aim was to explore the direction underlying the associations between ADHD symptoms and reading from middle childhood to early adolescence in order to test for predictive, potentially causal, relations. We expected bidirectional effects between ADHD symptoms and reading.

The second aim was to examine the genetic and environmental aetiologies of the association between ADHD symptoms and reading across this period. We hypothesized that there would be evidence for partial genetic overlap between ADHD symptoms and reading, and that many of the same genes would be involved in this association over time.

Finally, given the stronger phenotypic and genetic association of reading with inattentive than hyperactive-impulsive ADHD symptoms (Willcutt et al., 2000; Greven, Harlaar et al., 2011), we addressed aims one and two separately for inattentiveness and hyperactivity-impulsivity. We hypothesized that the longitudinal relationship with reading would be stronger for inattentiveness than hyperactivity-impulsivity.

Method

Participants and procedure

Data came from the Twins Early Development Study, a U.K. population-representative sample of twins born in England and Wales between 1994 and 1996 (Oliver & Plomin, 2007). Zygosity of twins was initially based on physical similarity ratings and later verified using genotyping (Freeman et al., 1997; Oliver & Plomin, 2007). Families were excluded from the present study following severe pre- or perinatal complications or if one or both twins suffered from a severe medical condition (e.g., autism, Down's syndrome). Uncertain sex or zygosity and failure to provide information at recruitment were also grounds for excluding families. After exclusions, a total of 1346 monozygotic (MZ) male, 1299 dizygotic (DZ) same-sex male, 1503 MZ female, 1360 DZ same-sex female and 2626 DZ opposite-sex twin pairs were included in the model fitting analyses.

ADHD symptoms and reading were assessed based on postal questionnaires, returned via postage-free envelope. Written consent, including consent to contact the twins' teachers, was obtained from parents at each assessment.

ADHD symptoms

ADHD symptoms (i.e. a total score of inattentive and hyperactivity-impulsive symptoms), inattentiveness and hyperactivity-impulsivity were assessed in middle childhood (mean age=7.87 years, SD=0.51) and early adolescence (mean age=11.34, SD=0.65). Ratings were

made using the DSM-IV-based ADHD items of the Revised Conners' Parent Rating Scale, which assesses hyperactivity-impulsivity and inattentiveness with 9 items each (Conners, Sitarenios, Parker, & Epstein, 1998). Parents rated each child's behavior on a 4-point Likert scale from (0) 'not true at all' to (3) 'very much true'. Cronbach's alphas were high: 0.91/0.91 for ADHD symptoms, 0.89/0.90 for inattentiveness and 0.84/0.83 for hyperactivity-impulsivity in middle childhood and early adolescence, respectively.

Reading

Reading ability was assessed in middle childhood (mean age=7.20, SD=0.28) and early adolescence (mean age=11.54, SD=0.66) based on U.K. National Curriculum criteria (National Curriculum, 2004). The National Curriculum specifies learning goals for 5- to 16-year-old children at state-funded schools in England and Wales. Teachers rated each child according to these learning goals, based on their knowledge of the child's performance over the past academic year (for further details see Chamorro-Premuzic, Harlaar, Greven, & Plomin, 2010; Kovas, Haworth, Dale, & Plomin, 2007). The National Curriculum teacher ratings demonstrate substantial agreement with reading tests (e.g., Dale, Harlaar, & Plomin, 2005).

The twin method

The twin method capitalizes on the fact that similarities between children raised together can be attributed to shared genetic or shared environmental factors. Genetic sharing is 100% for MZ (identical) twins, who share 100% of their inherited DNA sequence, but DZ (fraternal) twins on average only share 50% of their additive genes, like any other siblings. Shared environmental influences are 100% similar for MZ and DZ twins, and make children in the same family more alike. In contrast, non-shared environments, which contribute to differences between children in the same family, are specific to each child. Based on this, structural equation models of the expected covariation between twins can be constructed, which can be fitted to the observed data using maximum-likelihood iterative methods. This makes it possible to estimate the relative contributions of genetic (heritability A), shared environmental (C) and non-shared environmental (E) influences to individual differences in ADHD symptoms and reading and to the covariation between them.

The cross-lagged twin model

At the centre of the quantitative genetic cross-lagged model (first described by Burt, McGue, Krueger, & Iacono, 2005) are the stability paths (b_{11}, b_{22}), which connect the same traits across time, and the cross-lagged paths (b_{12}, b_{21}), which connect different traits across time (Figure 1). The stability and cross-lagged paths take the form of partial regression coefficients, which take into account the pre-existing association between ADHD symptoms and reading in middle childhood, as well as controlling for stability or cross-lagged effects. Figure 1 also depicts genetic (a_1–a_4), shared environmental (c_1–c_4) and non-shared environmental (e_1–e_4) contributions to ADHD symptoms and reading. Non-shared environmental contributions include measurement error. Lastly, Figure 1 presents genetic and environmental correlations (rA_1, rA_2; rC_1, rC_2; rE_1, rE_2). These can range from −1 to 1, and represent the extent to which genetic or environmental influences on the measures are shared.

Analyses

Standard regression-based corrections for sex and age were applied to raw scores (McGue & Bouchard, 1984) and residual scores were analysed. The ADHD measures were positively skewed and transformed using the command 'lnskew0' in STATA. Reading was normally distributed. Full-information maximum likelihood estimation was used, which allows the

inclusion of missing data with minimized bias (Finkbeiner, 1979). The significance of estimates was assessed using likelihood-based 95% confidence intervals (the inclusion of zero indicates non-significance). For the cross-lagged paths, significance was confirmed by fixing one cross-lagged path at a time to zero and assessing the drop in model fit using a chi-squared test (conclusions from confidence intervals were confirmed, although these chi-squared tests are not presented due to space limitations). Models were fitted to the data in Mx (Neale, Boker, Xie, & Maes, 2006).

Descriptive statistics, twin and cross-twin cross-trait correlations are available from previous publications based on the same data (Greven, Rijsdijk, & Plomin, 2011; Harlaar et al., 2007; Harlaar, Kovas, Petrill, Dale, & Plomin, 2011; McLoughlin, Ronald, Kuntsi, Asherson, & Plomin, 2007). In line with these publications, we modelled shared environmental influences and variance sex differences (the latter only for ADHD symptoms); however these publications provided no evidence for qualitative or quantitative sex differences, or sibling interaction.

Results

Phenotypic correlations

Phenotypic across-trait correlations between ADHD symptoms and reading ranged from −0.23 to −0.25, and were of similar magnitude within-age and across-age (Table 1). These correlations were negative because the measures were scored so that high scores refer to high ADHD symptoms and high reading ability. Phenotypic within-trait across-age correlations were 0.67 for ADHD symptoms and 0.48 for reading.

Model fit

The cross-lagged model of ADHD symptoms and reading (Figure 2) fitted the data well based on fit indices which heavily reward parsimony, as indicated by the negative values on the Bayesian Information Criterion and the Deviance Information Criterion (BIC=−586.18; DIC=−413.42). However, based on the significant χ^2 and positive Akaike's Information Criterion, the model provided inadequate fit compared to the baseline saturated model ($\chi^2(188)=520.36$, $p<.001$; AIC=144.36); although poor fit for these statistics is common for multivariate modelling in large samples.

Phenotypic cross-lagged model results

The stability paths pertaining to ADHD symptoms ($b_{11}=0.66$) and reading ($b_{22}=0.46$) were high and significant (Figure 2). Moreover, the cross-lagged paths from ADHD symptoms to reading ($b_{12}=-0.12$), and from reading to ADHD symptoms ($b_{21}=-0.07$) were negative and small, albeit significant. Of note, the cross-lagged path from ADHD symptoms to reading was significantly larger than the reverse path (confidence intervals did not overlap, and equating these paths resulted in a significant χ^2 difference test; $\chi^2(1)=58.80$, $p<.001$). This suggests that ADHD symptoms are a somewhat, albeit significantly, stronger predictor of reading than vice versa.

Genetic and environmental cross-lagged model results

Heritabilities in middle childhood were high for ADHD symptoms ($a_1^2=0.71$) and reading ($a_2^2=0.68$; Figure 2). The genetic correlation between ADHD symptoms and reading in middle childhood was modest ($rA_1=-0.30$), which suggests that ADHD symptoms shared around a third of genetic influences with reading (Figure 2). The genetic correlation was negative because the phenotypic correlation was negative. 85% of the phenotypic correlation between ADHD symptoms and reading in middle childhood was attributable to genetic influences (Table 2). This proportion can be derived from Figure 1 by calculating

[(a_1*rA_1*a_2)/phenotypic correlation in middle childhood]. In early adolescence, the same pattern was found: high heritabilities (a_3^2=0.67 for ADHD symptoms and a_4^2=0.54 for reading; Figure 2), moderate genetic overlap (rA_2=−0.29; Figure 2) and substantial genetic mediation of the phenotypic correlation between ADHD symptoms and reading (68%; Table 2). However, although the pattern of aetiologies is the same at the two ages, this does not necessarily mean that the same aetiological factors are involved in these traits and their association over time.

To address this issue, genetic and environmental variance in early adolescence was partitioned into variance specific to early adolescence and variance transmitted from middle childhood. First, variance can be transmitted via the stability paths, e.g., genetic variance transmitted via the stability path to ADHD symptoms is calculated as b_{11}^2*a_1^2 (Figure 1). Second, variance can be transmitted via the cross-lagged paths, e.g., genetic variance transmitted to ADHD symptoms via the cross-lagged path, is calculated as b_{21}^2*a_2^2. Third, variance can be transmitted via the covariation of ADHD symptoms and reading in middle childhood, e.g., genetic variance transmitted to ADHD symptoms via the covariation of ADHD symptoms and reading in middle childhood is calculated as 2*(b_{21}*a_2*rA_1*a_1*b_{11}). Results (summarized in online supplementary Table S1) show that for ADHD symptoms in early adolescence, 51% of genetic variance (i.e. of the heritability shown in Figure 2), 57% of shared environmental variance, and 65% of non-shared environmental variance was due to newly emerging aetiological influences specific to early adolescence. For reading in early adolescence, 67% of genetic variance, 84% of shared environmental variance, and 84% of non-shared environmental variance was attributable to newly emerging aetiological influences. Thus, more than half (51–84%) of genetic and environmental influences on ADHD symptoms or reading in early adolescence were different from those in middle childhood (the remaining 16–49% of genetic or environmental influences were transmitted from middle childhood).

Instead of partitioning variance in each trait, it was also possible to partition covariance between ADHD symptoms and reading into covariance specific to early adolescence and covariance shared with childhood. Out of the 68% of the phenotypic correlation between ADHD symptoms and reading in early adolescence due to genetic influences, a non-significant proportion of only 12% was due to newly emerging genetic influences specific to early adolescence, whereas the remaining 56% was due to enduring genetic influences transmitted from early adolescence (Table 2; 12% + 56% = 68%). This proportion can be derived from estimates in online supplementary Table S2, by calculating [($a_{3\ residual}$* $rA_{2\ residual}$* $a_{4\ residual}$)/phenotypic correlation in early adolescence)]=$\sqrt{0}$.34* −0.09*$\sqrt{0}$.36/−0.25 ≈ 12%. Thus, despite notable genetic innovation for each trait, the genetic factors involved in the *association* of ADHD symptoms and reading over time were highly stable.

Cross-lagged model results separately for inattentive and hyperactive-impulsive ADHD symptoms

The cross-lagged model was then fitted separately to inattentiveness and reading, and hyperactivity-impulsivity and reading in two bivariate models (see online supplementary Tables S3–S6; Figures S1–S2). As expected, phenotypic correlations with reading were significantly higher for inattentiveness (range from −0.25 to −0.29) than hyperactivity-impulsivity (−0.15 to −0.17; Table S3).

All cross-lagged paths between inattentiveness and reading (Figure S1), and between hyperactivity-impulsivity and reading (Figure S2) were significant, suggesting that these traits predicted each other over time. However, cross-lagged paths from inattentiveness to reading, and from hyperactivity-impulsivity to reading were significantly larger than the

reverse paths (χ^2 difference test equating cross-lagged paths in Figure S1: $\chi^2(1)=58.58$, p<.001; χ^2 difference test equating cross-lagged paths in Figure S2: $\chi^2(1)=26.08$, p<.001). This suggests that predictive relations were somewhat stronger in the direction from the ADHD symptom dimensions to reading. Notably, the cross-lagged path from inattentiveness to reading (standardized path estimate of −0.12) was significantly larger than the cross-lagged path from hyperactivity-impulsivity to reading (−0.08; confidence intervals did not overlap). This suggests that compared to hyperactivity-impulsivity, inattentiveness was a stronger predictor of reading.

Heritabilities were high for inattentiveness (0.78, 0.72) and hyperactivity-impulsivity (0.73, 0.67) in middle childhood and early adolescence, respectively (Figures S1–S2). At each age, reading showed significantly higher genetic correlations with inattentiveness (−0.42, −0.31) than with hyperactivity-impulsivity (−0.05, −0.13; Figures S1–S2). For hyperactivity-impulsivity the phenotypic correlations with reading were substantially mediated by shared environment (76% and 50% due to shared environment; Table S6). Phenotypic correlations between inattentiveness and reading were largely mediated genetically and negligibly by shared environment.

Of the 76% of the phenotypic correlation between inattentiveness and reading in early adolescence due to genetic influences, a non-significant proportion of only 12% was due to newly emerging genetic influences specific to adolescence, whereas the remaining 64% was due to enduring genetic influences transmitted from middle childhood (Tables S4, S6). For hyperactivity-impulsivity, out of 46% due to genes, the majority of this proportion was due to transmitted genetic effects (Tables S5–S6). Thus, genes involved in the association of reading with inattentiveness and hyperactivity-impulsivity were highly stable.

Lastly, a trivariate cross-lagged model of inattentiveness, hyperactivity-impulsivity and reading was fitted to the data (Figure S3). Analyses revealed that the cross-lagged path from hyperactivity-impulsivity to reading was significant in the trivariate model (standardized path estimate of −0.03; Figure S3). This suggests that hyperactivity-impulsivity predicts reading even after accounting for the strong phenotypic correlation between hyperactivity-impulsivity and inattentiveness in middle childhood (0.60; Table S3). Likewise, inattentiveness predicted reading independently of hyperactivity-impulsivity (standardized path estimate of −0.10; Figure S3). Of note, our bivariate analyses (Figures S1–2) were also important given the debate as to whether inattentiveness co-occuring with hyperactivity-impulsivity may be unrelated to inattentiveness without hyperactivity-impulsivity (Milich, Balentine, & Lynam, 2001).

Discussion

ADHD symptoms and reading showed reciprocal relationships across middle childhood to early adolescence. However, ADHD symptoms were a significantly stronger predictor of reading than vice versa. Inattentiveness and hyperactivity-impulsivity both contributed to the prediction of reading, although inattentiveness was a significantly stronger predictor of reading.

ADHD symptoms and reading were highly heritable and yielded no sex differences in their genetic and environmental aetiologies as shown previously for these data (Greven, Rijsdijk et al., 2011; Harlaar et al., 2007). The association between ADHD symptoms and reading was largely attributable to shared genetic influences. ADHD symptoms and reading each demonstrated notable genetic innovation; however the genes involved in their association over time were highly stable. This is consistent with a previous study (Ebejer et al., 2010), although we extend the result from middle childhood to early adolescence. As predicted,

reading showed significantly stronger genetic overlap with inattentiveness than hyperactivity-impulsivity. Genetics was largely responsible for associations between inattentiveness and reading, whereas genetic as well as shared environmental influences were important in explaining associations between hyperactivity-impulsivity and reading.

Limitations

One possible weakness of this study is its use of continuous ratings of ADHD symptoms in a population-based sample rather than focusing on the clinical extreme. However, the use of an unselected sample is also a strength as it facilitated the collection of data from a large sample, thereby increasing power, and helped avoid possible biases associated with clinical samples.

Furthermore, effect sizes of the cross-lagged paths were small. However, small cross-lagged paths are the rule rather than exception (e.g., Burt et al., 2005; Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009; Greven, Asherson, Rijsdijk, & Plomin, 2011; Hallett, Ronald, Rijsdijk, & Happe, 2010), and reflect the fact that the cross-lagged model is conservative, as cross-lagged paths are partial regression coefficients. The full predictive relationship between ADHD symptoms and reading is stronger, as indicated by the phenotypic correlations.

Moreover, to move from correlation to causation in cross-lagged analyses, synchronicity is required, that is, traits should be measured at the same time (Kenny, 1975). However, reading was assessed earlier than ADHD symptoms in middle childhood (7.2 versus 7.9 years), and somewhat later than ADHD symptoms in early adolescence (11.5 versus 11.3 years). In order to explore this possible bias, analyses in Figures 2 and S1–S3 were re-run after replacing the National Curriculum teacher ratings at age 7 with National Curriculum teacher ratings made at age 9 (mean child age=9.04, SD=0.29), more than one year after ADHD symptoms were assessed. Results (not shown) supported all conclusions drawn in the present paper.

An additional limitation is that the aetiologies underlying the cross-lagged effects are unclear, as cross-lagged paths are merely weighted by genetic and environmental influences (Luo, Haworth, & Plomin, 2010). For example, the cross-lagged path from ADHD symptoms to reading was attributable to genes and environments, exactly to the extent that ADHD symptoms in middle childhood were attributable to genes and environments.

Finally, limitations include standard limitations of the twin method (Plomin, DeFries, McClearn, & McGuffin, 2008) and of the cross-lagged model (Kenny, 1975), and the presence of statistically significant, albeit small, attrition biases linked to the presence of ADHD symptoms (Greven, Rijsdijk et al., 2011).

Implications

The significant and negative cross-lagged relations between ADHD symptoms and reading suggest that ADHD symptoms may put children at increased risk for reading problems, and vice versa. However, small effect sizes of cross-lagged paths suggest that the clinical implications of cross-lagged effects may be limited, at least for the developmental period under investigation. This is of interest as it suggests that although ADHD symptoms (especially inattentiveness) and reading significantly explain variance in each other over time, any predictive, and potentially causal, relations appear to be small.

Another result worth highlighting is that the association between hyperactivity-impulsivity and reading was substantially mediated by shared environment. This result replicates a previous finding based on reading assessed by tests rather than teacher ratings (Greven, Harlaar, et al., 2011). An obvious target for this shared environmental mediation is the

environments children share at home and school. Importantly, however, associations between hyperactivity-impulsivity and reading were only modest. Furthermore, additional evidence for this shared environmental mediation is needed, which may be complicated by several issues concerning shared environmental influences on ADHD (discussed in Burt, 2010).

A final implication concerns the finding that the genes involved in the association of ADHD symptoms and reading were highly stable across middle childhood to early adolescence. This was in spite of the relatively long follow-up into early adolescence and the large sample and hence increased power to detect newly emerging genetic influences. Moreover, this is by no means a default finding; for example newly emerging genetic influences have been shown to be almost as important as stable genes in the association of inattentiveness and hyperactivity-impulsivity over time (Greven et al., 2011). Findings from the present studysuggest that enduring molecular, and hence neurobiological, processes may be important for the association of ADHD symptoms and reading across childhood development. The exploration of these processes, for example through molecular genetic and neuropsychological studies, will be an interesting area for future research. Lastly, although speculative, our finding that the aetiologies of this association are largely the same in childhood and early adolescence, may suggest that similar treatments may be effective in alleviating this association across development.

Conclusion

ADHD symptoms and reading demonstrated reciprocal relationships, although all effects were small. Furthermore, genetic influences involved in the association of ADHD symptoms and reading were highly stable.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Study supported by grants from the U.K. Medical Research Council (G0901245; and previously G0500079), the U.S. National Institute of Child Health and Human Development (HD49861, HD44454, HD59215) and the European Commission Sixth Framework Programme (018696). We are grateful to Twins Early Development Study families.

Abbreviations

ADHD	attention deficit hyperactivity disorder
MZ	monozygotic
DZ	dizygotic

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Key points

- Associations between ADHD symptoms and reading have been attributed to shared genetic influences. The present study examined the stability and change of these genetic influences and the predictive relations underlying this association from middle childhood to early adolescence.
- ADHD symptoms and reading significantly predicted each other over time, although ADHD symptoms were a significantly stronger predictor of reading than vice versa.
- This suggests that ADHD symptoms may put children at increased risk for reading problems and vice versa, although all predictive relations were of small effect.
- Genetic influences involved in the association between ADHD symptoms and reading were highly stable over time.
- Thus, enduring molecular and hence neurobiological processes may be responsible for this association.

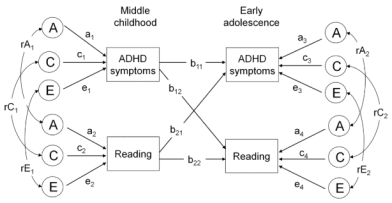

Figure 1.

Cross-lagged twin model. Rectangles represent observed variance in ADHD symptoms and reading. The circles labelled A, C, E represent latent genetic (A), shared environmental (C), and non-shared environmental (E) factors. Paths from the latent A, C, E factors to the observed variables (a_1–a_4, c_1–c_4, e_1–e_4) represent genetic and environmental contributions to ADHD symptoms and reading. Double-headed arrows (rA_1, rA_2, rC_1, rC_2, rE_1, rE_2) represent genetic and environmental correlations. Stability paths (b_{11}, b_{22}) connect the same traits across time. Cross-lagged paths (b_{12}, b_{21}) connect different traits across time.

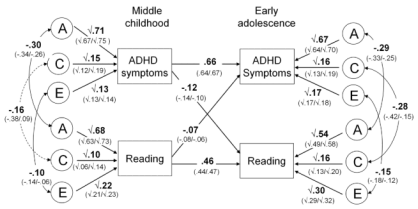

Figure 2.

Cross-lagged twin model results. For presentational purposes, A, C, E estimates in early adolescence refer to genetic and environmental contributions to total variance in ADHD symptoms and reading rather than contributions to residual variance specific to early adolescence. Path estimates are standardized. 95% confidence intervals are presented in parentheses. The dashed lines indicate the only non-significant path.

Table 1

Phenotypic correlations between ADHD symptoms and reading

		Middle childhood		Early adolescence	
		ADHD symptoms	**Reading**	**ADHD symptoms**	**Reading**
Middle childhood	ADHD symptoms	1			
	Reading	−.25 (−.26/−.23)	1		
Early adolescence	ADHD symptoms	.67 (.66/.68)	−.23 (−.25/−.22)	1	
	Reading	−.23 (−.25/−.22)	.48 (.47/.50)	−.25 (−.27/−.24)	1

Note: 95% confidence intervals in parentheses.

Table 2

Proportions of the phenotypic correlations due to genetic (A), shared environmental (C) and non-shared environmental (E) influences

	Middle childhood	Early adolescence	
	ADHD symptoms–reading	ADHD symptoms–reading	
r_p	−.25 (−.26/−.23)	−.25 (−.27/−.24)	
% of r_p due to			
A	.85 (.72/.98)	.68 (.58/.78)[a]	.12 (−.04/.27)[b]
C	.08 (−.04/.20)	.18 (.09/.27)[a]	.09 (−.04/.19)[b]
E	.07 (.04/.10)	.14 (.11/.17)[a]	.05 (.01/.09)[b]

Note: r_p = phenotypic correlation. 95% confidence intervals in parentheses.

[a] Proportions of the phenotypic correlation due to total genetic or environmental influences (i.e. transmitted plus age-specific effects).

[b] Proportions of the phenotypic correlation due to newly emerging genetic or environmental influences specific to early adolescence.